(FILE 'HOME' ENTERED AT 12:18:02 ON 10 JAN 2000)

FILE 'MEDLINE' ENTERED AT 12:20:07 ON 10 JAN 2000

L1	34920	S	CSF
L2	210	S	L1 (10A) INFLAMMATION
L3	20328	S	COLONY STIMULATING FACTOR
L4	34	S	L3 (10A) INFLAMMATION
L5	21	S	L3 (10A) (FORMYL OR F-MET?)
L6	3	S	L3 (3W) INFLAMMATION

L4 ANSWER 7 OF 34 MEDLINE
AN 1998323750 MEDLINE
DN 98323750
TI Castleman's disease and neutropenic enterocolitis presenting as Crohn's disease.
AU Burak K W; Bridges R J; Blahey W B
CS Department of Medicine, University of Calgary, Alberta.. burakk@cadvision.com
SO CANADIAN JOURNAL OF GASTROENTEROLOGY, (1998 May-Jun) 12 (4) 270-2. Journal code: CR9. ISSN: 0835-7900.
CY Canada
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199811
EW 19981103
AB A rare case of Castleman's disease presenting as Crohn's disease is described. This 21-year-old male with chronic neutropenia for one year presented with recurrent right lower quadrant pain of two years' duration. Small bowel follow-through suggested Crohn's of the terminal ileum. Colonoscopy confirmed ulcerations in the terminal ileum and cecum, with biopsies showing necrosis and **inflammation**. Treatment was initiated with prednisone, 5-aminosalicylate and granulocyte **colony-stimulating factor** for neutropenia. Symptoms recurred one year later, and repeat colonoscopy showed a focal cecal ulceration. Two years after presentation a resection was planned. Laparotomy revealed a normal ileocecal region and a large retroperitoneal mass of lymphadenopathy. Biopsies confirmed reactive hyperplasia, consistent with the plasma cell variant of Castleman's disease. Chemotherapy has resulted in improvement of symptoms and decrease in mass size, but cecal ulceration persisted. This case illustrates a variant presentation of Castleman's disease with neutropenia and manifestations in the gastrointestinal tract.

L7 ANSWER 3 OF 5 MEDLINE
AN 96315736 MEDLINE
DN 96315736
TI CD4+ T cell inducible immunoregulatory cytokine response in rheumatoid arthritis.
AU al-Janadi N; al-Dalaan A; al-Balla S; Raziuddin S
CS Department of Medicine, College of Medicine, King Saud University, Abha, Saudi Arabia.
SO JOURNAL OF RHEUMATOLOGY, (1996 May) 23 (5) 809-14.
Journal code: JWX. ISSN: 0315-162X.
CY Canada
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199612
AB OBJECTIVE. Monocytes and CD4+/CD8+ T cells produce immunoregulatory cytokines that participate in the pathogenesis of various immune disorders. We investigated the secretion of Th1-Th2 cell response cytokine production of CD4+/CD8+ T cells from the synovial fluid (SF) and blood of patients with rheumatoid arthritis (RA). METHODS. Blood and SF purified monocytes, CD4+ and CD8+ T cells were stimulated with bacterial lipopolysaccharides or anti-CD3 antibody, and secretion of various cytokines was determined by bioassay or ELISA. RESULTS. Monocytes from SF and blood of patients with RA produced highly elevated levels of interleukin-1 alpha (IL-1 alpha), IL-6, tumor necrosis factor-alpha (TNF-alpha), and granulocyte macrophage **colony stimulating factor** (GMCSF), the leading mediators of **inflammation**. However, CD4+ T cells secreted deficient levels of IL-2 and interferon-gamma (IFN-gamma), but higher levels of IL-4 and IL-10, the typical immunoregulatory Th2 cell response cytokines. CD8+ T cells also produce elevated levels of IL-4 and IL-10 but almost normal levels of IFN-gamma in this disease. CONCLUSION. The cytokine produced by monocytes (IL-alpha, IL-6, TNF-alpha, and GMCSF) and by CD4+ T cells Th2 cell responses (IL-4 and IL-10) may exert immunopathologic and immunoregulatory effects in SF and thus mediate some of the clinical

L5 ANSWER 16 OF 21 MEDLINE
AN 92112688 MEDLINE
DN 92112688
TI Nuclear signaling in human neutrophils. Stimulation of RNA synthesis is a response to a limited number of proinflammatory agonists.
AU Beaulieu A D; Paquin R; Rathanaswami P; McColl S R
CS Le Centre de Recherche en Inflammation, Immunologie et Rhumatologie du Centre de Recherche du Centre Hospitalier de l'Universite Laval, Quebec, Canada.
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1992 Jan 5) 267 (1) 426-32.
Journal code: HIV. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199204
AB At inflammatory sites, neutrophils are stimulated by a range of proinflammatory molecules which elicit a number of cellular responses. Considerable information on the cytoplasmic events that occur following activation of neutrophils at the cell membrane level already exists. In this study, we have focused on the ability of neutrophil agonists to initiate nuclear signaling events by investigating the induction of de novo RNA synthesis. Of a total of 14 different known potent leukocyte agonists, only three had a significant effect on the induction of RNA synthesis in neutrophils; the formylated oligopeptide **formyl** -methionyl-leucylphenylalanine (fMet-Leu-Phe), granulocyte-macrophage **colony-stimulating factor**, and tumor necrosis factor alpha. All three agonists induced de novo RNA synthesis in neutrophils at concentrations known to be optimal for the activation of a number of other cellular responses occurring in inflammation. Of significance was the observation that activation of RNA synthesis in neutrophils is a G-protein-mediated event, is also dependent on tyrosine phosphorylation, but is not influenced by cAMP. Finally, we have demonstrated that all three agonists also induce de novo synthesis of a limited number of proteins, with granulocyte-macrophage colony-stimulating factor and fMet-Leu-Phe having the most potent effect. These studies define the effects of neutrophil agonists on de novo RNA and protein synthesis in a proinflammatory context and suggest that these events in neutrophils occur in a restricted fashion, highly dependent on the stimuli

L5 ANSWER 18 OF 21 MEDLINE
AN 91217640 MEDLINE
DN 91217640
TI Activation of the adhesive capacity of CR3 on neutrophils by endotoxin: dependence on lipopolysaccharide binding protein and CD14.
AU Wright S D; Ramos R A; Hermanowski-Vosatka A; Rockwell P; Detmers P A
CS Laboratory of Cellular Physiology and Immunology, Rockefeller University, New York, New York 10021..
NC AI-22003 (NIAID)
AI-24775 (NIAID)
GM-40791 (NIGMS)
SO JOURNAL OF EXPERIMENTAL MEDICINE, (1991 May 1) 173 (5) 1281-6.
Journal code: I2V. ISSN: 0022-1007.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199108
AB Tumor necrosis factor alpha, granulocyte **colony-stimulating factor**, granulocyte/macrophage **colony-stimulating factor**, and **formyl** peptide were each found to cause a twofold increase in expression of CD14 on the surface of polymorphonuclear leukocytes (PMN). Upregulation of CD14 was complete by 20 min and thus appeared to result from expression of preformed stores of protein. The CD14 on the surface of PMN was shown to serve two biological functions. It bound particles coated with complexes of lipopolysaccharide (LPS) and LPS binding protein (LBP). This binding activity was enhanced by agonists that upregulated CD14 expression and may serve in the clearance of Gram-negative bacteria opsonized with LBP. Interaction of CD14 with LPS in the presence of LBP or serum also caused a dramatic, transient increase in the adhesive activity of CR3 (CD11b/CD18) on PMN. Enhanced activity of CR3 and other members of the CD11/CD18 family underlies many of the known physiological responses of PMN to LPS and may be a central feature of the in vivo responses of PMN to endotoxin.

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Symptoms recurred one year later, and repeat colonoscopy showed a focal cecal ulceration. Two years after presentation a resection was planned. Laparotomy revealed a normal ileocecal region and a large retroperitoneal mass of lymphadenopathy. Biopsies confirmed reactive hyperplasia, consistent with the plasma cell variant of Castleman's disease. Chemotherapy has resulted in improvement of symptoms and decrease in mass size, but cecal ulceration persisted. This case illustrates a variant presentation of Castleman's disease with neutropenia and manifestations in the gastrointestinal tract.

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